Bandolier 160

Independent evidence-based thinking about health care

Plus çe change

One of the main stimuli for evidence-based medicine all those years ago was an understanding that much of what was published in the medical literature was wrong. In 1991 Richard Smith, then editor of the BMJ, quoted from an article by David Eddy "*...only 1% of the articles in medical journals are scientifically sound*" [1].

Since then much has been done to try and rectify matters, especially the tremendous work done by the international panels that have worked on CONSORT, QUOROM, and other statements about how randomised trials, systematic reviews, diagnostic test and observational studies should be reported. There is guidance for health economic papers too. Yet anyone who reviews for journals or reads extensively could be forgiven for thinking that there had been no change. Barely two years ago Ioaniddes could write a paper entitled "Most published research findings are false" ([2]; Bandolier 139).

Over a decade ago someone suggested that Bandolier should cease because we all knew about evidence, and that from then on evidence would be used properly to make healthcare decisions. Actually, we ran out of steam and resources, losing our puff after ranting about evidence for a decade and a half. So this month, for a bit of fun, we show how to construct an argument based on limited evidence, and by breaking the rules.

Bandolier Internet

To reiterate what was said in Bandolier 159, the Internet version will continue. It is likely to change somewhat over the coming year as we formulate new ideas.

References:

1 R. Smith quoting Prof. D. Eddy. BMJ 1991 303: 798-99.
2 J Ioannides. PLoS Medicine 2005 2: e124.

In this issue

On supporting belief

Bandolier is occasionally allowed out to attend the odd meeting or two. And some are very odd indeed, especially when it comes to the use of evidence.

There is the wrong way. This is characterised by finding some piece of evidence that backs up whatever opinion is already held. Ideally, something from a randomised trial, or even a meta-analysis does the trick, especially with some incomprehensible output, like an odds ratio portrayed in a forest plot. But of course, it's always then possible to come out with that immortal phrase "the evidence shows…." It helps, of course, to do this with swaggering confidence.

Then there is the right way, which, is always a bit more tricky. This involves setting some standards as to what constitutes good evidence, and holding up what evidence there is to that standard. Evidence that fails to meet the standard can be discarded, or at least used with caution. Even evidence that does meet the standard may even then not tell the whole story, perhaps being informative about efficacy, but not effectiveness, or being incomplete in some other way.

In a nutshell, then, trying to do things the right way can make anyone look like a hesitant fool compared to the confident assurance of someone who just wants to win the argument. Bandolier has chosen a current controversy, that of cardiovascular problems with NSAIDs and coxibs, as an example of how to win an argument, if not be right.

Selecting some data

If one thing is true, it is that over the last decade research into clinical effects of cyclooxygenase-2 inhibitors has produced a huge amount of information from randomised trials and observational studies. Most comes from patients with pain, but some comes from exploratory work in precancerous conditions or dementia.

For the purposes of this exercise we will choose the following:

- To use only large randomised trials that compare coxib with NSAIDs. This means the CLASS, VIGOR, TARGET and MEDAL trials for celecoxib, rofecoxib, lumiracoxib, and etoricoxib in patients with osteoarthritis and rheumatoid arthritis, generally aged in their 60s and 70s. Doses of coxibs (celecoxib 800 mg/day, rofecoxib 50 mg/day, lumiracoxib 400 mg, and etoricoxib 60 mg or 90 mg daily) were generally above those indicated for these conditions in clinical practice.

- To use only myocardial infarction as an outcome. While several different cardiovascular endpoints have been chosen, like APTC or all thrombovascular events, myocardial infarctions comprise the majority.
- To analyse by the number of patients exposed.
- To analyses by the number of patient years of exposure.
- To analyse according to whether patients were using or not using low dose aspirin (LDA).
- To analyse according to naproxen or other NSAIDs as comparator(s).

The information was obtained from the published papers, or other commentaries that included the data in the form required. Information from the MEDAL programme was provide by Merck Inc.

The raw data

Table 1 shows the raw data used for the analyses, with data for CLASS and TARGET separated by different comparators, which were diclofenac, naproxen, and ibuprofen. Average duration of therapy was derived from information for all patients, and may be slightly different for aspirin or non-aspirin populations, and varied from about a third of a year for CLASS to 1.5 years for MEDAL.

The number of events was small for most of the trials, a product of low event rates (about 0.5% a year for myocardial infarction in the age group in the trials, and relatively short duration. Only the MEDAL programme, because of its combination of size and duration, had more than 30 MIs in total. Of course, when split by use or non-use of LDA, the numbers of events is smaller still, which increases the possible effects of chance. This is important if the aim of the exercise is to find arguments to use for or against the proposition that coxibs cause heart attacks.

Results

Table 2 has the results for calculations based on aspirin status, by the number of patients, or the number of patient years of exposure, and by comparison with all NSAIDs and non-naproxen NSAIDs. Figures 1-2 show the L'Abbé plots for the comparison with all NSAIDs by number of patients, for all patients (Figure 1), those not taking LDA (Figure 2) and those taking aspirin (Figure 3).

What is interesting about the Figures is that there is a tendency for the larger studies to be smack on the line of identity, while the smaller ones, most likely influenced by the random play of chance, are found away from the line of identity.

Table 1: Numbers of myocardial infarctions, patients, and patient years of exposure for coxibs and NSAIDs in large randomised trials, for all patients and by low dose aspirin status

Trial	Coxib	NSAID	Number of MIs	Number of patients	Patient years of exposure	Number of MIs	Number of patients	Patient years of exposure	Average years on treatment	
All patients										
			Coxib			NSAID			Coxib	NSAID
Class	Celecoxib	Ibuprofen	7	1997	722	7	1985	690	0.36	0.35
Class	Celecoxib	Diclofenac	11	1990	719	4	1996	694	0.36	0.35
Vigor	Rofecoxib	Naproxen	20	4047	3035	4	4029	3022	0.75	0.75
Target	Lumiracoxib	Ibuprofen	5	4376	3326	7	4397	3034	0.76	0.69
Target	Lumiracoxib	Naproxen	18	4741	3651	10	4730	3595	0.77	0.76
Medal	Etoricoxib	Diclofenac	111	16833	25858	122	16504	24797	1.54	1.50
Patients not taking low dose aspirin										
			Coxib			NSAID			Coxib	NSAID
Class	Celecoxib	Ibuprofen	6	1574	569	2	1567	545	0.36	0.35
Class	Celecoxib	Diclofenac	7	1580	571	2	1602	557	0.36	0.35
Vigor	Rofecoxib	Naproxen	20	4047	3035	4	4029	3022	0.75	0.75
Target	Lumiracoxib	Ibuprofen	4	3401	2585	5	3431	2367	0.76	0.69
Target	Lumiracoxib	Naproxen	10	3549	2733	4	3537	2688	0.77	0.76
Medal	Etoricoxib	Diclofenac	65	11104	17057	61	10918	16404	1.54	1.50
Patients who were taking low dose aspirin										
			Coxib			NSAID			Coxib	NSAID
Class	Celecoxib	Ibuprofen	1	423	153	5	418	145	0.36	0.35
Class	Celecoxib	Diclofenac	4	410	148	2	394	137	0.36	0.35
Vigor	Rofecoxib	Naproxen	0	0	0	0	0	0	0.75	0.75
Target	Lumiracoxib	Ibuprofen	1	975	741	2	966	667	0.76	0.69
Target	Lumiracoxib	Naproxen	8	1192	918	6	1193	907	0.77	0.76
Medal	Etoricoxib	Diclofenac	46	5729	8801	61	5586	8393	1.54	1.50

Patients taking LDA had a higher risk of myocardial infarction than those not taking LDA, by a factor of at least two-fold, as might be expected, so there is an argument that analysis by aspirin status is a legitimate and sensible thing to do.

For almost all the various sub-group analyses undertaken, there was no statistical difference, with the confidence interval of the relative risk including 1. In those taking LDA, the relative risk was invariably below 0.8.

In those patients not taking LDA there was a statistical increase, whether the results were analysed by number of patients or by number of patient years. The relative risk in both cases was 1.4, with the lower confidence interval approaching 1. The NNH was about 800.

Argument for a link between coxibs and MI

It's staring you in the face. In those folk not taking LDA there was a statistically significant increase in MI in people taking coxibs. What more do you want? For every thousand people you give a coxib to, one is going to have a heart attack. And they are being given out like sweeties. It is a potential public health disaster.

*Just because people have a little pain? Give me a break! Surely this isn't worth **any** risk.*

Argument against a link between coxibs and MI

All you get is bare statistical significance for one sub-group, of one set of trials, and for one outcome? And not for the comparison with non-naproxen NSAIDs, with overall results influenced by naproxen? Maybe you should wonder whether there is a real effect at all, or perhaps whether naproxen is the only one of your drugs reducing MIs. Why is there no effect in people at greater risk of a heart attack, but only in those at lower risk? Can you explain that? Is there a biologically plausible mechanism for such a situation?

Anyway, overall there is no effect statistically, even with coxib doses generally above those used clinically. Any difference is clinically insignificant and probably unmeasurable even if it were there. It is always useful to have several different therapies available because of the large inter-individual differences between patients, whose genetic reasons we are just beginning to understand. And chronic pain is miserable, with huge negative impact on quality of life. So don't worry.

Table 2: Analysis based on the number of patients, or patient years of exposure, by aspirin status for coxibs compared to all NSAIDs and non-naproxen NSAIDs

A: Calculations based on the number of patients

	MI/Total (N)		MI/Total (%)			
Patient group	**Coxib**	**NSAID**	**Coxib**	**NSAID**	**Relative risk (95% CI)**	**NNH (95% CI)**
Comparison with all NSAIDs combined						
All patients	172/33984	154/33641	0.51	0.46	1.1 (0.89 to 1.4)	not calculated
Taking LDA	60/8729	76/8557	0.69	0.89	0.77 (0.55 to 1.1)	not calculated
Not taking LDA	112/25255	78/25084	0.44	0.31	1.4 (1.07 to 1.9)	750 (420 to 3900)
Comparison with only non-naproxen NSAIDs						
All patients	134/25196	140/24882	0.53	0.56	0.94 (0.74 to 1.2)	not calculated
Taking LDA	52/7537	70/7364	0.69	0.95	0.72 (0.50 to 1.03)	not calculated
Not taking LDA	82/17659	70/17518	0.46	0.40	1.2 (0.84 to 1.6)	not calculated

B: Calculations based on the number of patient years of exposure

	MI/Total (patient years)		MI/Total (% per year)			
Patient group	**Coxib**	**NSAID**	**Coxib**	**NSAID**	**Relative risk (95% CI)**	**NNH (95% CI)**
Comparison with all NSAIDs combined						
All patients	172/37311	154/35832	0.46	0.43	1.1 (0.86 to 1.3)	not calculated
Taking LDA	60/10761	76/10248	0.56	0.74	0.75 (0.54 to 1.05)	not calculated
Not taking LDA	112/26550	78/25583	0.42	0.30	1.4 (1.03 to 1.8)	850 (450 to 7200)
Comparison with only non-naproxen NSAIDs						
All patients	134/30625	140/29215	0.44	0.48	0.92 (0.73 to 1.2)	not calculated
Taking LDA	52/9843	70/9342	0.53	0.75	0.71 (0.49 to 1.02)	not calculated
Not taking LDA	82/20782	70/19873	0.39	0.35	1.1 (0.81 to 1.54)	not calculated

Figure 1: Individual comparisons of MI rates with coxibs and NSAIDs (solid symbols naproxen) - all patients

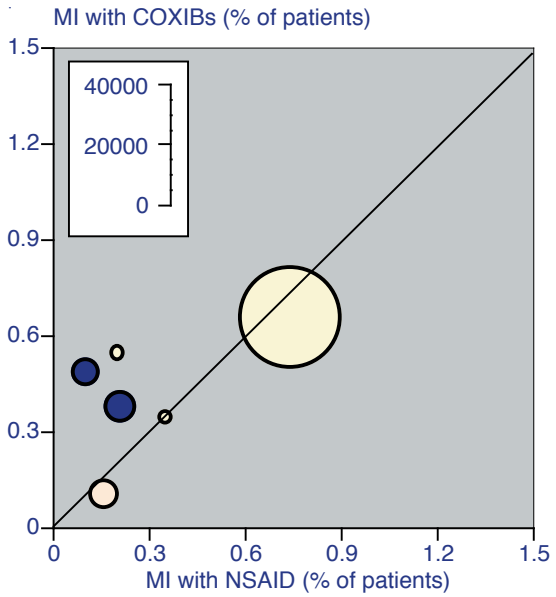


Figure 2: MI rates with coxibs and NSAIDs - patients not taking LDA

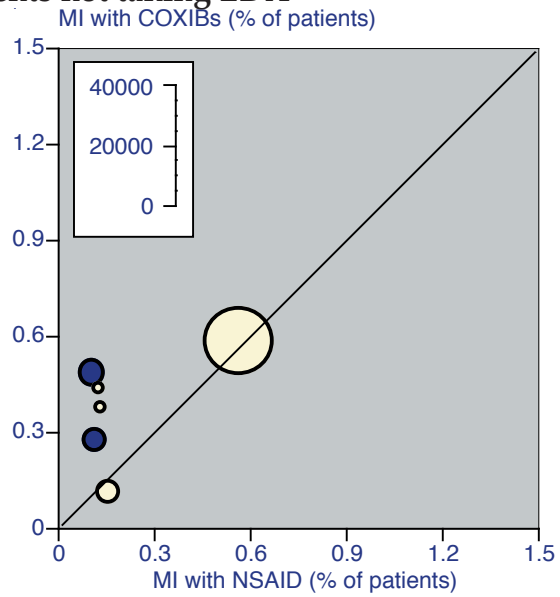
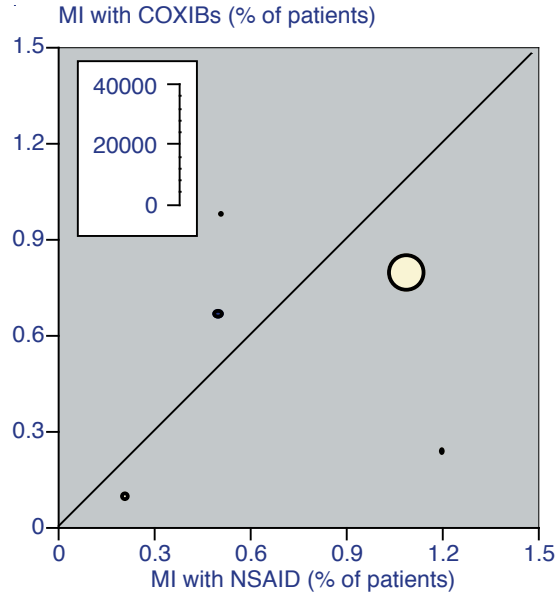


Figure 3: MI rates with coxibs and NSAIDs - patients taking LDA



Comment

Let's reiterate here that this exercise is to demonstrate how opposing views might be generated from the same data, chosen because there was an overall sufficiency of events from large randomised trials in which the MIs were judged by independent blinded endpoint committees.

All of us feel the need to question data, to ask about whether bigger or smaller effects occur in those at higher risk, or on different drugs, or who wear blue or are Welsh. Such salami slicing is disapproved of when used to find a positive result in a trial because of the loss of statistical power that results in turning a randomised trial, in effect, into an observational study with good data collection in a well-defined population.

What's the right answer? Curiously, with the amount of information available, it remains a tad elusive. As more information becomes available, the weight appears to be on the side of no increased risk from coxibs at standard doses, or at least nothing substantial. The trouble is that we know less about NSAIDs than the coxibs; we only know anything sensible about NSAIDs because of their inclusion in coxib trials.

And yet

There's always an "and yet". Two simple thoughts stand out.

Firstly, that MI rates seem to be lower with naproxen than other NSAIDs, as the Figures show, at least within the context of regular long term dosing in a clinical trial.

Second, the evidence is clear of higher rates of myocardial harm for coxibs and possibly aspirin compared with placebo in trials in precancerous conditions, though emphatically not in dementia. The former is characterised by low annual event rates with placebo, with rates less than a quarter of those in the latter.

There are some signals where cardiovascular risk is low – but that may just be another case of reading too much into the data. In any event, it is interesting to see the results from these trials and try and relate them to current guidance.

And finally

And finally, this is nothing like the whole story in the arguments about coxibs, NSAIDs, and cardiovascular events. It isn't even a partial story. All it is is an attempt from one set of data to show how "evidence" can be organised to support two sides, or any sides, of an argument.

What we have to do is forget, if we can, our prior beliefs, and judge the evidence in front of us. That includes issues around quality, size, and validity, as well as issues like the weight we give to multiple comparisons without any statistical adjustment for those multiple comparisons. The Figures suggest, once again, that the big battalions might be the ones that don't speak with forked tongue.

Gastrooesophageal reflux and BMI

There is a general understanding of a relationship between weight and increased prevalence of heartburn, or symptoms of gastrointestinal reflux. Indeed, there is a meta-analysis [1] indicating significant increase for those with a BMI of 25 kg/sq m or more compared with those with lower BMI.

However, this is something of a blunt analysis, and does not tell us much about gradations. For instance, is there a gradual increase in risk, or does the risk increase dramatically at any particular BMI? Is there any evidence of a U-shaped relationship, perhaps with higher rates in underweight people? A new study [2] fills in some of the fine details.

Study

Part of the US Nurses study, this survey involved a questionnaire to a random selection of 12,192 nurses, with questions about frequency, severity, and duration of heartburn or acid regurgitation, using validated definitions of both terms. Severity was defined as mild (can be ignored), moderate (cannot be ignored but does not affect lifestyle), severe (affects lifestyle), and very severe (markedly affects lifestyle). Frequent was an episode occurring at least weekly.

Information was collected regarding height, weight (at various ages), drugs, diet, exercise, tobacco and alcohol use, and concurrent disease. Analysis of results used these data to examine confounding variables. Controls were women without symptoms not taking acid suppressing medicines.

Results

The women in the survey had an average age of 66 years, and an average BMI of about 27. Women with symptoms were more likely to have a higher BMI, use medications for asthma or hypertension, or hormone preparations, consumed more calories, and were less active.

Over 10,500 questionnaires were returned, with an 86% response rate. No symptoms of heartburn or acid reflux were reported in 41% (1 in 10 of whom were using proton pump inhibitors), with the remaining 51% reporting symptoms less frequently than monthly, to daily (Figure 1). One woman in five (22%) had symptoms at least weekly. Of those with symptoms, most (95%) were moderate or mild, and only about 5% had symptoms that were severe or very severe, and most (55%) had both heartburn and acid reflux.

Figure 1: Frequency of symptoms

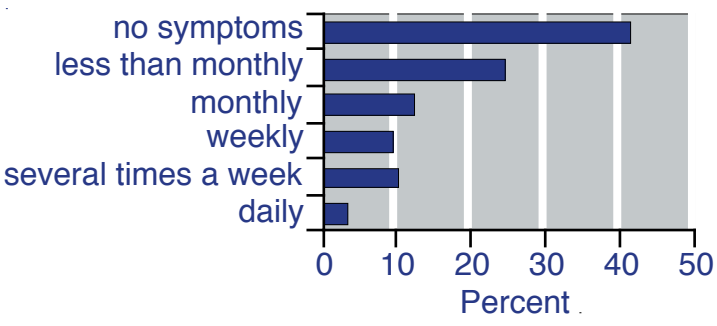
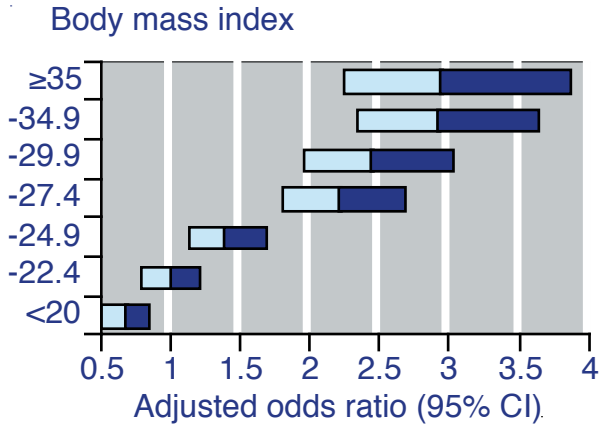


Figure 2: Odds ratio for heartburn or acid reflux at different BMI levels



Using those women with frequent (at least weekly) symptoms, and women without symptoms as controls, there was increasing reporting of symptoms of heartburn or acid reflux with increasing BMI (Figure 2), even after adjusting for potential confounders. This was the case for mild, moderate and severe or very severe symptoms. With a BMI ≥25, 60% of the increased risk was accounted for by excess weight.

Among women who had gained weight during the previous 14 years, there was a dose-dependent increase in the risk of symptoms, with about a threefold increase in those whose BMI increased by 3.5 units. Conversely, there was a reduction in almost 40% in the risk of frequent symptoms in women who reduced BMI by 3.5 units or more.

Comment

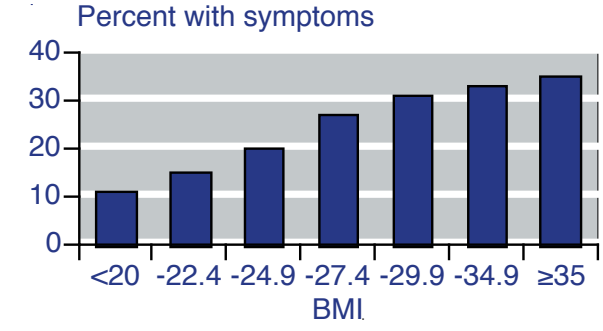
This nicely captures the relationship between increased risk of heartburn or acid reflux and increased weight. Being or becoming overweight doubles the risk of having these symptoms at least once a week. A back-of-the-envelope calculation gives crude results for the prevalence of moderate or severe heartburn or acid reflux symptoms at least weekly for each band of BMI and shows the gradation (Figure 3). The bottom line is that this is yet another reason to avoid being overweight, along with all the others. If our populations keep growing out as well as in numbers, we will need to step up production of the antacids.

References:

1 H Hempel et al. Meta-analysis: obesity and the risk for gastroesophageal reflux disease and its complications. Annals of Internal Medicine 2005 143: 199-211.

2 BC Jacobson et al. Body-mass index and symptoms of gastroesophageal reflux in women. New England Journal of Medicine 2006 354: 2340-2348.

Figure 3: Crude symptom rate and BMI



Fracture and quality of life in older women

Fractures in older people, especially older women, can be problematical. The impact of hip fracture can be devastating. Much treasure is spent on trying to prevent fracture through treatment of osteoporosis, and by trying to reduce loss of bone, especially in postmenopausal women. If we want to know how treatments compete in the clever world of cost effectiveness, then we have to measure the negative impact of fractures, and while much has been done in that area, a new, and very large, study [1] opens another window.

Study

This was part of a prospective longitudinal study of 200,000 postmenopausal US women aged at least 50 years, without a diagnosis of osteoporosis, no bone density measurement within 12 months, and not taking treatments for osteoporosis. For inclusion they has to have completed two mail or telephone surveys, the first at about 12 months after enrolment, and the second about 36 months after enrolment.

Both surveys elicited information of new fractures, health status using a SF-12 instrument, osteoporosis-related care, and fall history. Analysis of the SF-12 data was according to two composite scores, the physical component score (PCS) and mental component score (MCS). Reported new fractures (hip, spine, wrist, rib) between the first and second surveys formed the cases, with controls being women without fracture.

Results

The analysis included 86,128 women (88% white), whose mean age was about 65 years. Just 1.2% had suffered a fracture in the year before the first survey. Fractures between the first and second survey numbered 320 hip, 445 spine, 835 wrist, and 657 rib, 2.6% over the two years. There were 83,871 women without fracture who served as controls.

Women suffering a fracture more frequently had significantly reduced bone mineral density, and were 4-6 times more likely to have suffered a fracture during the 12 months before the first survey. They also had lower quality of life scores at the first survey.

After adjusting quality of life scores for these factors, women suffering a fracture in the two years between the two surveys had significantly reduced PCS scores compared with women without a fracture (Figure 1). Statistically significant reductions were found for hip, spine, wrist and rib fractures for younger postmenopausal women (50-64 years), and for hip, spine and rib fractures in older postmenopausal women (65-99 years).

Women suffering a fracture in the two years between the two surveys had greater reduction in MCS scores than women without a fracture (Figure 2). Statistically significant reductions were found for spine and rib fractures for younger postmenopausal women, and for hip and spine fractures in older postmenopausal women.

Comment

What makes this study worth thinking about is the combination of its size and detail, and that it provides quality of life results for different fractures in younger and older postmenopausal women. It also did some useful statistical stuff, like taking into account multiple comparisons, so that statistical significance was only reported when the probability value was 0.004, so it does not tell us about associations that crept into conventional levels of significance.

Those in the know about such things may not have been surprised by the findings. For the rest of us, perhaps what stands out is the particular loss of life quality attendant on vertebral fractures. This may reflect the fact that vertebral fractures cannot be healed, and often come with a lot of back pain, and we do know that chronic pain has a large negative impact on quality of life.

It also helps to have some context. The negative impact on PCS scores for hip and spine were at the same level as those for COPD, hip impairment, or rheumatoid or osteoarthritis. Given that we will have more older people with low bone

Figure 1: Reduction of physical quality of life compared with control

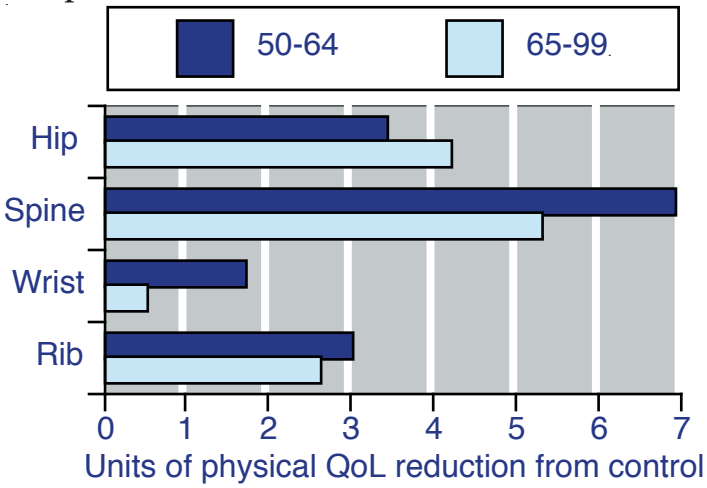
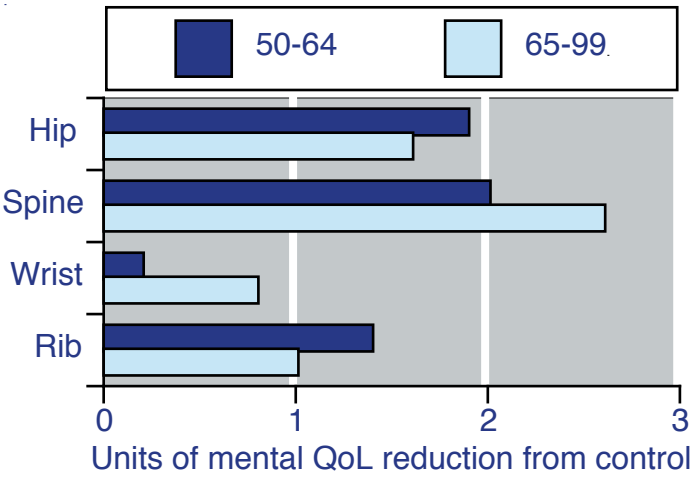


Figure 2: Reduction of mental quality of life compared with control



mineral density and at risk of these fractures, this should help in making sense of current and new therapy choices.

Reference:

1 SK Brenneman et al. Impact of recent fracture on health-related quality of life in postmenopausal women. Journal of Bone and Mineral Research 2006 21: 809-816.

Statins, sepsis, and chronic kidney disease

Bandolier once came a cross a paper that claimed that at least half of all indications for drug use arose from observations made by perceptive clinicians, rather than from the original intentions for their use by pharmaceutical companies. It is interesting, therefore, to perhaps see one swim into our ken, and perhaps watch it develop. The case of the possible effect of statins in reducing sepsis may be one of these.

Study

A prospective observational study [1] has examined the use of statins and rate of sepsis in dialysis patients. Situated in the USA, the study began in 1995 to examine treatment choices and outcomes. Eligibility included long-term outpatient dialysis in the preceding three months in adults of at least 17 years, and it enrolled 1041 participants up to mid-1998, with observations continuing up to 2005.

Statin use was determined by review of clinic notes and computerised records. Data collected was extensive, including demographics, comorbidity, drug therapy, and laboratory values. The primary outcome was hospital admission for sepsis, where sepsis was defined using ICD codes. A number of different statistical analyses were performed, including multivariate regression and propensity score matching.

Results

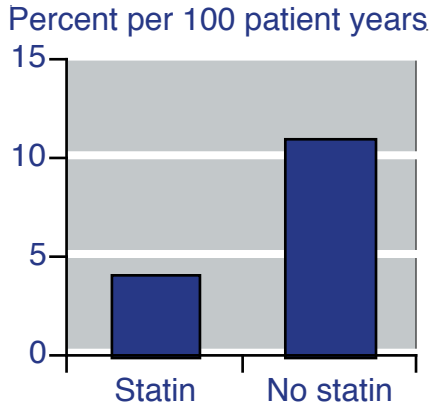
The mean age of patients was 57 years, about half men, and about 80% white. Statin users were more likely to be white, and have higher cholesterol levels, cardiovascular disease, and a history of sepsis, but were less likely to have used street drugs, and consumed less alcohol.

In the 1041 patients there were 303 hospital admissions for sepsis over the mean follow up of 3.4 years. The crude incidence rate was 4% per year in statin users and 11% per year in non-users (Figure 1). In the main statistical analysis, the crude incidence rate ratio was 0.37 (95% confidence interval 0.22 to 0.61). Using multivariate analysis with more complex interaction models, or propensity scoring, did not reduce the effect, but if anything made it larger. Various sensitivity analyses did not change the findings.

Comment

This was an extremely detailed study, with a moderate number of events, and with extensive efforts to discover possible sources of confounding, especially confounding

Figure 1: Crude rate of hospital admission for sepsis with and without statin



by indication. It found none of these, and the result, a 60% reduction in the risk of sepsis with statins in dialysis patients looks strong.

Several other observational studies in bacteraemia or bacterial infection have also found improved outcomes in statin users, and a study of hospital admission for cardiovascular events found a lower incidence of sepsis with statin use. Moreover, there appears to be a biological plausibility, as the first statin was originally identified from a penicillin fungus, where it is theorised that it may have benefited the fungus by preventing replication of microorganisms requiring cholesterol for growth.

All in all an intriguing story based on some good observation. It will be interesting to see where it leads.

Reference:

1 R Gupta et al. Statin use and hospitalization for sepsis in patients with chronic kidney disease. JAMA 2007 297: 1455-1464.

Waiting for cataracts

Cataracts are common in older people. There are more older people, with a huge expansion in the over-65 population predicted in western countries over the next 20-50 years. Something like eight out of 10 people over the age of 75 years will develop cataracts within a 10 year period. For providers of healthcare, cataract surgery is a major issue, and planning for provision stimulates the government neurone.

Whatever the benefits of cataract surgery, one of the issues that causes great controversy is the amount of time someone has to wait for it. After all, six months, or a year, may be a large percentage of time available to someone in their 70s or 80s, especially when impaired vision makes life increasingly difficult. A systematic review [1] looks at the evidence about the consequences of delay.

Systematic review

The review examined a large number of electronic databases and searched conference abstracts for studies that might throw light on the question. There were sensible limitations

on studies allowed. For instance, studies had to be relatively modern, reflecting current procedures, have a suitable diagnostic method or examination, and relate to industrialised countries (it was specifically Canadian in its focus). The aim was to examine the visual, adverse event, and quality of life relationships for waiting time for cataract surgery.

Results

The search found two randomised trials, three prospective cohort studies, and 22 descriptive studies.

Visual

No studies examined postoperative visual outcomes, though the decline in visual acuity during the course of the wait was documented in many of the studies.

Adverse events

One randomised study in the UK noted that people who waited had increased rate of falls and more fractures (12% of them had a fracture) compared with expedited surgery, where the fracture rate was 3%.

Quality of life

A number of studies found that quality of life was reduced in people who waited for cataract surgery, with increased dissatisfaction when the wait was longer than about three months.

Comment

Patients who wait six months or more for cataract surgery experience negative outcomes during the wait period, including vision loss, reduced quality of life, and falls. Managers might ask for evidence about what constitutes an acceptable wait. Given that waiting begins when a patient experiences symptoms sufficient to warrant a GP visit and referral, that seems to be a useful time to start the clock.

While there is little evidence that, say, three months is worse than two months, there seems no good reason to set the barrier at longer than six weeks.

Reference:

1 W Hodge et al. The consequences of waiting for cataract surgery: a systematic review. Canadian Medical Association Journal 2007 176: 1285-1290.

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Book review

Christopher Newdick. Who should we treat? Rights, Rationing, and Resources in the NHS. Oxford University Press 2005. ISBN 0-19-926418-X. 270 pp. £29.95.

Faced with a bleak Bank Holiday weekend, there's nothing that bestirs the blood better than to settle down to read a book about the UK National Health Service written by a lawyer from a legal point of view. Imagine, a glass of ginger beer, some sherbet lemons, and you're set. Or maybe you'd just prefer to go hang gliding.

Well, the exciting choice may just be the book. Unlikely, yes, but Newdick's approach mixes a bit of fun with information and stimulus. For instance, where would you start to try and understand the NHS, assuming you had just arrived from Mars? Not any old government waffle, or party political broadcast, or tabloid hype. No, being from Mars and very logical, you would ask for a copy of the law that set up the NHS and it's governance.

Perhaps that is where trouble starts. The NHS is organised principally under an act of Parliament of 1977, but has been in a state of almost permanent revolution since 1980. Each subsequent act has made changes to the 1977 act, or some other act; new acts change previous acts, and newer acts change those, with the consequence that there is no single piece of paper that tells you what it is like now. So our friend form Mars would do well to have several pairs of arms, a large pair of scissors, and plenty of time to cut and paste.

There is no single place for ordinary mortals to get the single piece of paper saying how the NHS should be run, and for all practical purposes the rights and duties arising in connection with the NHS are inaccessible. Only when you know how the system should run, how it does run, and some of the ethical principles of how resources should be allocated, can you begin to consider thinking about whom to treat.

In the book there's tons of interesting stuff about making difficult decisions, written in a fairly racy style with lots of references to appropriate sources, be the Hansard, the DoH, NICE, or legal cases that can so affect how society works. And there are examples, especially highlighting the problem of variation, and how different conclusions may be reached, and actions taken, depending on where you start from and the particular circumstances you are in. Some nice evidence, especially the effect of NICE approval for herceptin around the country. While the percentage of eligible cancer patients receiving herceptin pre-NICE was almost always below 20%, 12-18 month afterwards in varied between 90% and below 10%, in a pretty linear way - making for an interesting take on the postcode lottery that NICE was supposed to end.

If you want to work in the NHS and are not retiring too soon, or you want to cause trouble, or sell to the NHS, or even if you have an intellectual need to have a challenge, this is the book for you. Stock up on the ginger beer and pray for a wet bank holiday.